

**Amendment to the specification:**

Please delete the Abstract on Page 57, lines 1-37, in its entirety and replace with the following amended Abstract of Disclosure:

ABSTRACT OF DISCLOSURE

A compound having a binding affinity for the cholecystokinin (CCK)-receptor, in particular a derivatized peptide having a binding affinity for the CCK-B-receptor and useful for binding or detecting CCK-B-receptors in tumors is described. The compound comprises a CCK-8 peptide or CCK-8 peptide analog capable of binding the CCK-receptor that is labelled with a metal isotope or atom by means of a chelating agent coupled to the peptide. This compound is useful for diagnosing and supporting therapy of tumors that express CCK-B receptors.

PATENT

Please delete the Paragraph [0109] on Page 19 in its entirety and replace with the following amended Paragraph [0109]:

[0109] The FIG. 1 attached shows displacement curves of [ $^{125}$ I]-CCK-10 analog (compound 15) binding to tissue sections from three different tumours: A=medullary thyroid carcinoma (MTC) and B=small cell lung carcinoma (SCLC) and C=Gastro entero pancreatic tumour (GEP-Tu).

Please delete the Paragraph [0110] on Page 19 in its entirety and replace with the following amended Paragraph [0110]:

[0110] Tissue sections are incubated with 20,000 cpm/100  $\mu$ l [ $^{125}$ I]-CCK-10 and increasing concentrations of unlabelled CCK-8 (unsulfated)( $\blacktriangle$ ), CCK-8 (sulfated) ( $\bullet$ ) or ~~somastotatin~~ somatostatin (SS-14) ( $\circ$ ). Each point represents the optical density of binding measured in the tumour area. Non-specific binding is subtracted from all values. In all cases, complete displacement of the ligand is achieved by sulfated CCK and unsulfated CCK is inactive in GEP-TU, whereas somastotatin is inactive in the nanomolar range for all three types of tumours.

Please delete the Paragraph [0113] on Page 20 in its entirety and replace with the following amended Paragraph [0113]:

[0113] FIG. 2 attached shows displacement curves of [ $^{125}$ I]-CCK-10 (compound 15) binding to tissues from two different tumours: A=medullary thyroid carcinoma (MTC) and B=Meningioma of different DTPA-substituted unsulfated CCK-8. Tissue sections are incubated with 20,000 cpm/100  $\mu$ l [ $^{125}$ I]-CCK-10 and increasing concentrations of compound 16 (CCK-8 (sulfated))(●), compound 19 (■), compound 20 ( $\Delta$ ), compound 21 ( $\diamond$ ), compound 22 ( $\blacktriangle$ ), compound 23 ( $\square$ ) and compound 24( $\circ$ ).

Please delete the Paragraph [0117] on Page 20 in its entirety and replace with the following amended Paragraph [0117]:

[0117] FIG. 3 attached shows displacement curves of [ $^{125}$ I]-CCK-10 (compound 15) binding to tissues from medullary thyroid carcinoma (MTC) of two different  $^{115}$ In-DTPA substituted desulfated CCK-8 analogs. Tissue sections are incubated with 20,000 cpm/100  $\mu$ l [ $^{125}$ I]-CCK-10 and increasing concentrations of compound 16 (CCK-8 (sulfated))(●), compound 25 ( $\blacktriangle$ ) and compound 26 (■). Each point represents the optical density of binding measured in the tumour area. Non-specific binding is subtracted from all values. In all cases, complete displacement of the ligand is achieved.

Please delete the Paragraph [0120] on Page 21 in its entirety and replace with the following amended Paragraph [0120]:

[0120] The FIG. 4 attached shows displacement curves of [ $^{125}$ I]-desulfated-CCK-10 binding (compound 13 or 14) to tissue sections from medullary thyroid carcinoma (MTC). Tissue sections are incubated with 20,000 cpm/100  $\mu$ l [ $^{125}$ I]-desulfated-CCK-10 and increasing concentrations of compound 17 (CCK-8 (unsulfated)) ( $\blacktriangle$ ), compound 16 (CCK-8 (sulfated)) ( $\bullet$ ), CCK-10 (unsulfated) ( $\blacktriangledown$ ) or ~~somastotatin~~-somatostatin (SS-14) ( $\circ$ ). Each point represents the optical density of binding measured in the tumour area. Non-specific binding is subtracted from all values. In all cases, complete displacement of the ligand is achieved by sulfated and unsulfated CCK, whereas somastotatin is inactive in the nanomolar range. The two different mono  $^{125}$ I-iodinated compounds appear to have the same affinity. FIG. 5 attached shows the autoradiogram of the binding of  $^{125}$ I desulfated CCK-10 ligand to CCK-B receptors in MTC. A= Autoradiogram showing total binding of the ligand; B= Autoradiogram showing non-specific binding (in the presence of  $10^{-6}$  desulfated CCK-10 analog).